Whole Food Nutrition and Chronic Degenerative Disease

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The last fifty years have been both exciting and revolutionary in nutritional research. Among our many discoveries, we have learned how complex and intricate the inner workings of the human body are. These developments have helped us understand our environment and how our body interacts with it. Although this understanding has grown in leaps and bounds, there still remains a vast amount of unknown processes yet to be discovered. We have only begun to understand and grasp the complexity that surrounds us. In many ways, the historically reductionistic framework of research makes understanding these intricate processes rather difficult and labour-intensive. Yet it is this very research that has lead to the understanding that no single constituent is responsible for the antioxidant benefit against the generation of chronic disease obtainable from a whole food diet.

In 2004 the two most common causes of death from chronic degenerative disease in Canada included Cancer (30%) and Heart disease (23%) (Statistics Canada 2008). Combined, these account for more than half of deaths in Canada -- explicating the need for research and development in chronic disease etiology, progression and prevention. What we have learned in the past fifty years is that oxidative stress is largely responsible for the damage resulting in various chronic diseases. Using cancer progression as an example -- the stages of development include initiation, conversion, propagation and metastasis. Both oxidative damage and immune system dysfunction mediate carcinogenesis and no single event is responsible in cancer etiology but rather multiple insults and accumulation of these abnormalities are required to provoke carcinogenesis.

Likewise we have discovered the importance of a whole food, fruit and vegetable based diet in the protection against such disease. Fruits and vegetables contain various antioxidants in the form of polyphenols, flavones which are protective against oxidative damage. In 2007 a prospective cohort investigation by the European Prospective Investigation into cancer and nutrition revealed an inverse relationship between mortality and the intake of fruiting vegetables, root vegetables and fresh fruit (Agudo et al 2007). These results are consistent with several other studies from around the globe, notably Lock et al concluded in the Bulletin of the World Health Organization that the low consumption of fruits and vegetables is estimated to attribute to 2.6 million deaths each year (Lock et al 2005). A modest intake of 600g of fruits and vegetables per day was estimated to reduce coronary vascular disease by 31%, lung cancer by 20%, stomach and esophageal cancer by 20% (Lock et al 2005).

Prospective cohort studies had overwhelmingly convinced researchers that a fruit and vegetable based diet was protective against chronic degenerative disease, yet the active constituents remained a mystery. Markers such as Vitamin C, E and beta carotene had all been used as a measure of fruit and vegetable intake in these prospective studies. Stahelin et al (1991) noted a 60% higher cancer risk in the lowest versus highest quintile of serum beta carotene levels. Other researchers found similar trends with all markers of fruit and vegetable intake and in their haste to explain the antioxidant activity they confused correlation with causation. Intervention studies soon eluded that Beta Carotene, Vitamin E and Vitamin C as supplements were not the most active constituents in fruits and vegetables. The Lancet published an article on a randomized placebo controlled trial with the intervention of all three of these nutrients and found the incidence of coronary heart disease to be the same in both groups (Heart Protection Group 1996). Jacob et al (2003) noted no change in markers of oxidant damage in men with low fruit and vegetable intake and moderate supplementation of these compounds. Some studies even suggested that intervention with these essential nutrients (Vitamin E, Vitamin C and Beta Carotene) could indeed increase the risk of these chronic degenerative diseases. The Beta Carotene Cancer Prevention Study Group (1994) found an 18% increase in lung cancer incidence and 8% mortality rate from intervention with Beta Carotene.

The reductionistic assumption that correlation meant causation became apparent. Yet even today some prospective papers still note Vitamin C, E and beta carotene as the causative agents. Their remains a certain amount of heterogeneity in the findings regarding the above constituents and the quality of the intervention supplement have often been criticized. Although there are many therapeutic uses for these agents they do not appear to be the key constituents in fruit and vegetable intake that offer a protective effect against chronic degenerative disease. New strategies were then developed to discern oxidant/antioxidant relationships within the body. Soobrattee et al. (2005) used five different in vitro strategies (TEAC,FRAP,HOCl, deoxyribose assay and Cu-phen assay) in analyzing the various antioxidant power of phenolic compounds commonly found in fruit and vegetables. These antioxidant capacity tests are widely discouraged in isolation as the mechanisms of antioxidant reactions within the human body are quite complex. Soobrattee et al (2005) discovered that many of these compounds had higher scores than Vitamin C and other reference compounds hinting at the active constituents. Procyanadins, Epigallocatechin gallate (EGCG) and Quercetin had TEAC values of 7.58, 4.39, 3.68 respectively – where as Vitamin C offers only a 1.42 TEAC. All the compounds measured also appeared to excel in a certain specialty of antioxidant capacity – reinforcing the importance of a varied whole food diet. It is estimated that the total antioxidant power of an apple is composed primarily

by these smaller components (eg. Flavanoids and phenols) and vitamin C is only responsible for 0.4% of its antioxidant power (Liu 2003).

The understanding of oxidant damage and its role in chronic degenerative disease remained and Prieme et al (1997) summed up these observations by concluding that " the cancer protective effects of fruits and vegetables seems to rely not on the effect of single antioxidants but rather on other anti-carcinogenic compounds or on a concerted action of several micronutrients present in these foods". This course of evidence necessitates the need for nutrient rich, varied whole food diet to achieve the antioxidant health promoting effects from food. A small study involving 23 healthy subjects was able to demonstrate decreased number of endogenous DNA strand breaks in lymphocytes (a marker of oxidant damage) simply by supplementing with tomato juice, carrot juice and dried spinach powder over two-weeks (Pool-Zobel et al 1997). Several other small studies with relatively poor design have found varied results (Meller et al 2003) and to date no long-term intervention trial has been conducted. Methods of measuring oxidant damage, sample size and the quality of intervention tend to be the greatest limitations to these studies examining fruit and vegetable supplementation on incidence of oxidant damage. We can however assume from the literature to date that a whole food diet rich in a variety of antioxidant compounds, both essential and non-essential, is the key to prevention of chronic degenerative disease. The inner workings of the human body most certainly are complex and intricate; good health hinges on our ability to nourish each and every one of these intricate processes.

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